UNITED STATES DISTRICT COURT
WESTERN DISTRICT OF OKLAHOMA
Case No. CIV-14-665-F

RICHARD GLOSSIP, et al.,
Plaintiffs,
vs.
RANDY CHANDLER, et al.,
Defendants.

REMOTE VIDEOTAPED DEPOSITION OF
DR. JOSEPH ANTOGNINI
January 28, 2021
10:03 a.m. EST

Reported by:
Debra Stevens, RPR-CRR

January 28, 2021

10:03 a.m. EST

Remote Videotaped Deposition of Dr. Joseph Antognini, held at the above date and time, before Debra Stevens, RPR/CRR and Notary Public of the State of New York.

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Also Present:
LEE BOWRY, Videographer

E X A M I N A T I O N S

E X H I B I T S

THE VIDEOGRAPHER: Good morning. We are going on the record at 10:03 a.m. on January 28, 2021. Please note that audio and video recording will continue to take place unless all parties agree to go off the record. This is media unit 1 of the video-recorded remote virtual deposition of Dr. Joseph F. Antognini in the matter of Richard Glossip, et al., versus Randy Chandler, et al., filed in the United States District Court, Western District of Oklahoma, Civil Action No. CIV-14-665-F.

My name is Lee Bowry from the firm of Winter Reporting, a Veritext company, and I am the videographer. The court reporter is Debra Stevens, also with Winter Reporting. I am not authorized to administer an oath. I am not related to any party in this action, nor am I financially interested in the outcome.

Counsel attending remotely will

now state their appearances and affiliations for the record. If there are any objections to proceeding please state them at the time of your appearance, beginning with the noticing attorney.

MR. STRONSKI: This is Jim Stronski. I am from the law firm Crowell & Moring and we represent Plaintiffs in this case. With me is Pilar Stillwater, also with the firm Crowell & Moring; and other co-counsel will introduce themselves.

MR. LIEBERMAN: Good morning. This is Michael Lieberman from the Federal Public Defender Capital Habeas Unit in the Western District of Oklahoma. Also appearing but not on screen, Alex Kursman, K-U-R-S-M-A-N, who is also an assistant federal defender. He is with the Community Defender Office in the Eastern District of Pennsylvania; and Kim Stout, S-T-O-U-T, who is also an

assistant federal public defender with the Federal Public Defender in District of Arizona.

MS. KOLODINSKY: This is Lynne Kolodinsky. I recently changed my name. It is K-O-L-O-D-I-N-S-K-Y.

MS. STILLWATER: Were you able to hear me?

MR. MANSINGHANI: This is Mithun Mansinghani for the Defendants from the Oklahoma Office of the Attorney General.

THE VIDEOGRAPHER: Will the reporter please swear the witness.

COURT REPORTER: If you would state your full name?

THE WITNESS: Joseph Francis Antognini.

Whereupon,

DR. JOSEPH F. ANTOGNINI,

having been first duly sworn/affirmed, was examined and testified as follows:

THE VIDEOGRAPHER: We may proceed.

DR. J. ANTOGNINI

EXAMINATION BY

MR. STRONSKI:

Q. I am Jim Stronski. I introduced myself before. Good morning, Dr. Antognini.

A. Good morning.

Q. Are you aware of any study that used midazolam to induce and maintain general anesthesia in the face of surgical stimuli?

A. I presume you are asking about this in humans. Is that correct?

Q. Correct.

A. And when you -- could you repeat the question? I want to make sure I understood. Induce and maintain?

Q. Yes. I am interested in any study that reliably used midazolam as the only drug to induce and maintain general anesthesia for purposes of surgery.

A. Well, surgery -- let me just address the surgery part. Surgery -- there are different types of surgery, different types of surgical stimuli and

noxious stimuli. Certainly midazolam has been used as a sole drug to induce anesthesia for a procedure, as I have mentioned in my report.

(Reporter interruption.)

A. As I mentioned in my report, there are -- I did cite studies where midazolam has been used to induce anesthesia in preparation for endotracheal intubation, which is very stimulating. And that, as I said, is in my report.

One of the challenges of using midazolam to maintain anesthesia would be that you need a very large dose to be able to achieve that, so large that nobody has actually attempted to do that for a prolonged surgical procedure.

So, if we are talking about a very short procedure, there are studies, I think, that indicate that, that you could use midazolam for painful procedures, otherwise painful procedures. But for prolonged procedures, no, there are no studies in humans where midazolam has been

used alone for the purposes I mentioned because of the massive dose that would likely need to be administered.

Q. But in humans, to use your words, such a massive dose has not been studied clinically. Correct?

A. That is correct, to my knowledge.

Q. So, you have no basis in science, in data, to opine that midazolam at any dose would maintain anesthesia. Correct?

A. I do not have any -- again, there are no data that I am aware of, any published studies where midazolam has been used by itself for a prolonged surgical procedure. By that I mean for hours and hours. So, that is correct. And that is because the dose that would be required or to even study that would be so large that it wouldn't be ethically or clinically worthwhile to pursue.

Q. So you rely upon the Gehrke reference. You mentioned endotracheal

intubation. That was your example. Am I right in remembering that?

A. That particular study, you mean?

Q. Yes. I asked you if there are studies where midazolam was used in humans to induce and maintain anesthesia for surgery, and the example I think you gave me was endotracheal intubation. Is that right?

A. That is correct. There is more than the Gehrke study, though, that I have cited.

Q. Are there any other procedures -- how long does the endotracheal intubation take? What is the range of time it takes?

A. The actual intubation involves what is called laryngoscopy, basically where you actually take a metal tongue blade, as I tell patients, and insert it into the mouth, open up the airway. And then you actually place the endotracheal tube. All that might take on average a minute, a minute and a half.

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But it's not just the procedure itself that is stimulating. You have that large plastic tube into the trachea or the windpipe, and it's there after you are done, so it's still stimulating. In my report, I talked about the Miyake study, where they had given midazolam for induction. They also gave other drugs. Both drugs wore off very quickly and those patients were basically lying there with this endotracheal tube for about 60 minutes, and that's stimulating. The authors, as I recall, talk about that.

So, it's not just the procedure itself. It's actually the tube that is there. Since we all know, have experienced aspiration, where something went down the wrong way and you have coughed violently, well, that is what that tube is like, and it's there basically past the procedure itself. So, that also is stimulating.

Q. How long does the tube stay there?

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A. It depends on the particular procedure. It could be there for days after surgery. We can leave the tube in or it can come out after surgery, 30 minutes after, an hour, two hours. Depends on the length of the surgery.

Q. So there is no cutting done with the endotracheal intubation, right? That is not the intent of it; correct?

A. No. There is no cutting done, but it is certainly more stimulating in terms of anesthetic requirements than a skin incision.

Q. So, is it your testimony in Gehrke and in the other Miyake reference that you know that those patients were receiving no other drugs?

A. Well, both the Miyake study and the Gehrke study that I have cited -- so the Gehrke study, there was a subgroup of patients that did not receive other drugs, at least according to their report. Some of them did receive opiates. But the conclusion was that midazolam was still a

good drug to use in the study, even the study of midazolam by itself. If they recognized midazolam by itself was not, adequate, they would have mentioned that, I would think, and they did not. The other --

Q. Did they say it was adequate?

A. That is my recollection. We can certainly pull up the reference, but that is --

Q. We'll look at it.

A. That's my recollection of it.

As far as the Miyake study is concerned, those patients received an opiate called remifentanil with the induction, and then the remifentanil was discontinued right afterwards. And that is a drug that -- its effects dissipate very, very quickly. After five, ten minutes or so or something like that, maybe shorter, the effects are gone.

So, that's one of the reasons why, if you look at how these patients behaved in terms of their

electroencephalogram, it's pretty clear that 20, 30 minutes out or more, the remifentanil is gone and that basically these patients are doing okay.

Also in the discussion section, as I recall -- we will have to pull it up. In the discussion section of the Miyake study, they talked about essentially a preliminary study, and I believe there they did not use remifentanil. So again -- and they got more or less the same result. So, that leads me to believe and opine that midazolam is sufficient to anesthetize patients for the endotracheal intubation and for the continued placement or presence of that endotracheal tube.

Q. In these cases, you said that the actual intubation takes a minute or so. Did you say that? A minute, minute and a half?

A. Yes. It can be shorter if it's an easy airway and you are skilled, or it can be a little longer. You know, somewhere around there is my guess.

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Q. Could be 30 seconds?

A. Could be 30 seconds, yes.

Q. Then it is just there for however long and then it's removed at some point. Is that fair?

A. That is correct, yes.

Q. And it could be there, you said, for days?

A. If the patient, after surgery, needs to be in an intensive care unit and needs a respirator or ventilator, yes, it could be there for days.

Q. So is it your testimony that these patients in the Miyake and Gehrke study had their tubes in for an extended period of time without any other medication but for midazolam?

A. Well, for the Miyake study that is correct. They stopped the study basically at 60 minutes. After that they basically continued on with surgery and they gave more anesthetic at that point. I don't recall exactly what they used. For the Gehrke study, I don't recall what

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happened afterwards.

But in general, patients that have endotracheal tube in place in the intensive care unit, they require some type of sedation or anesthesia or something, some drug, because it is very stimulating. Virtually all of the hospitals that I am aware of have some type of protocol to provide some types of drugs, including midazolam. That's a very common drug to be used as an infusion in intensive care unit so that these patients will tolerate that endotracheal tube. It is pretty unusual for a patient to not require some type of drug, medication such as midazolam or something like that -- propofol is sometimes used -- to ensure that they can tolerate that tube.

Q. Are you familiar with the continuum of depth of sedation definitions of general anesthesia and levels of sedation, analgesia that have been approved by the ASA Committee of Delegates?

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milliequivalent level, which is your experience in administering potassium chloride, the low range of that experience, if you compare it to the amount administered in the Ohio lethal injection protocol, that amount is 24 times greater --

MR. MANSINGHANI: Object to form -- sorry.

Q. Do you have any knowledge of any studies that would characterize the nature of the kind of pain that results administering 24 times the amount of a drug that is known to cause pain at 10 milligrams milliequivalents?

MR. MANSINGHANI: Object to form.

A. There are no studies that I am aware of that would quantify that. And my experience with potassium chloride has been in the operating room primarily. Again, I have had patients in the recovery room -- that is not the term we use anymore but that is what most people will

recognize -- and then also in the ICU.

Most of my own personal professional experience with potassium chloride has been in the operating room. Of course, you have to infuse it slowly because if you give it too fast you can have a cardiac arrest. That is the main reason why it is infused slowly.

But in that setting, in my experience, in anesthetized patients there is no indication that the patients are experiencing any type of response to it because I don't recall there being increases in the heart rate or blood pressure or anything else when potassium is being infused.

Q. How many times do you recall infusing potassium during anesthesia?

A. I can't give you a number. It is going to be maybe, you know, a dozen, two dozen times maybe. I just don't -- you are talking about a 30-year career more or less. I can't remember.

Q. So, it is not something

controlled.

Q. Similarly, the osmolarity of injectable drugs is adjusted so that you don't have a hyper or hypo osmolar substance in the blood or in the veins. Correct?

A. That is often done, yes. There is adjustment of the osmolarity.

Q. And that is because it is well understood that either can cause damage and pain on injection. Correct?

MR. MANSINGHANI: Object to form.

A. I don't know that people have systematically studied the amount of pain that occurs with alterations in osmolarity. So, that is a little bit -- I am not aware of anything -- that information may exist. I just don't know about it.

Q. Okay. But it is done because it can damage tissue, whether or not that causes -- the pain it causes we'll put aside, but it damages the tissue.

Correct?

A. The experience over many years is that veins can be damaged by drugs that are going through them. So, often that is damage that occurs over the course of a long time, not just from one injection but over the course of many hours to days. So, it is also time dependent.

Q. And the potassium chloride also, at 240 milliequivalents, will depolarize the cells and trigger the nerve fibers in the veins. Correct?

A. That is my understanding of that area. I don't have any more specifics as to what the mechanism would be.

Q. And that would be experienced as severe pain unless you put the brain stem to sleep and had a state of general anesthesia. Correct?

MR. MANSINGHANI: Object to form.

A. The presence of a drug, an anesthetic drug or a drug that affects the brain will reduce the experience of

somebody who's receiving potassium chloride. And I have opined that midazolam is such a drug, especially at the dose used in the protocol, that it would drastically alter any experience of the usual pain that we think about that would occur from potassium chloride.

Q. Is it your opinion that midazolam puts the brain stem to sleep at this dose?

MR. MANSINGHANI: Object to form.

A. I don't really typically think of using terminology "putting the brain stem to sleep." Midazolam will affect the -- have effects in the brain. It has effects in the brain stem, it has effects in the spinal cord. So, that drug, midazolam, at that dose we are talking about, 500 milligrams, would drastically reduce the experience someone would have when they are awake? The answer is yes. It would be a combination of effects at sites.

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in these dosages, that midazolam and diazepam are of similar potency?

A. In this setting -- let's see here. Let me just look closely at these figures and their data here.

I have no reason to basically refute what they have reported. Is it surprising? Again, I am not surprised in the sense that, you know, sometimes you get results that you sometimes don't think you will. Are these data different from what others have reported? Possibly. So, I will just state that I guess.

Q. Let's go to the Miyake reference, which you also relied upon and mentioned, which is Exhibit 853.

(So marked for identification as Exhibit 853.)

Q. If we go to the first page, is this the Miyake reference that you referenced earlier?

A. Yes, it is.

Q. What do you rely upon it for?

A. So, this study was performed to

look at effects of midazolam on the electroencephalogram primarily. And they were looking at dose response effects. They gave .2 milligrams per kilogram in one group and .3 milligrams per kilogram in the other group.

The important thing that I looked at here is that these patients were given the midazolam, they were intubated with muscle relaxant and also an opiate that was very short lived. The remifentanil drug wears off very quickly.

So, they were intubated and then left basically on a ventilator over the course of 60 minutes. And during that time where basically all they had lingering around, so to speak, was the midazolam, the remifentanil has worn off and the patients were paralyzed, to use the term, a paralytic with vecuronium. These authors reported no change in the BIS basically over that 60-minute period.

Then in the discussion section, the second-to-last paragraph, actually

starting on the left-hand side, which is going to be page 392 of their paper, or 443 on the bottom of this exhibit, they talk about a preliminary study and --

Q. Can I just stop you a second. Where are you talking about the preliminary study? It's on 39 --

A. 392, towards the bottom of the left-hand column.

Q. Okay. Thank you.

A. So in this preliminary study they basically did the same type of study, although it only went out, it looks like, to 20 minutes. They used the same doses and found -- and this is without the remifentanil -- and found that the BG data, the BIS data, did not on average change over the course of that period, indicating again that the presence of the stimulating tube and being on the ventilator was not sufficient to push the BIS number up from the low 60's. What that indicates to me is these patients were not waking up because the BIS number was

staying pretty stable there in the low 60's.

Again, the combination of the earlier data or the data they reported in the study going out to 60 minutes and then the preliminary study that they report here indicates that these patients were not waking up from the fact that they were paralyzed, and it was because they had received that midazolam.

Q. I am looking for where the description of the preliminary study begins. I am sorry but I am not finding it. Where would I look? I am on page 392.

A. So the paragraph -- scroll down a little bit. Where it says, just like any paper will say, "There are several limitations to our study." So, you go there and they go through some of the limitations. And they want to talk about the fact that they did use remifentanil for the intubation period for these patients. And they were looking at not so

much what was happening with the intubation but what was going to be happening afterwards in terms of the patient's electroencephalogram and their BIS level after that. So, they wanted to let the remifentanil wear off in their study, which is what they did.

But just to rule that out altogether, that remifentanil was not a factor in what they saw, they had a preliminary study where they didn't use remifentanil at all and did not see a change basically -- once the electroencephalogram was depressed by the midazolam, it stayed depressed despite the fact that these patients were on the ventilator and with an endotracheal tube.

Q. Okay, I see where we are now. So the preliminary study, if you go down on that page, has an N equals 12, mean age 59 plus or minus 11 years. Is that the preliminary data?

A. Yes, that is what they are referring to. So then they also state

that "these data are comparable with those reported here with remifentanil, suggesting that the infusion of remifentanil unlikely affected the EEG data."

Q. Basically you have 96 plus or minus 2; 64, et cetera. Those are the BIS numbers. The plus or minus is some deviation based on the 12. Is that correct?

A. That's correct.

Q. What is SEF95?

A. That is another EEG measure. That stands for spectral edge frequency 95. It's just another EEG measure.

Q. Now, the initial study involved 5,000 patients, right, the study that is the subject of this paper? Is that correct?

A. No, no. Not nearly 5,000 patients, no.

Q. It says, "A prospective cohort study involving approximately 5,000 consecutive surgical patients revealed --"

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oh, that is a different study.

So, there are 12 in the preliminary study. How many subjects are there in the study that is the subject of this paper?

A. There were 12 subjects in the group that received 0.2 milligrams per kilogram of the midazolam and there were 12 subjects in the 0.3-milligram per kilogram group.

Q. That also both received the remifentanil?

A. Correct.

Q. How much fentanyl -- how much midazolam did the preliminary group get?

A. They state it's 0.2 or 0.3 milligrams per kilogram. N equals 12, but they don't specify how many of the 12 received 0.2 and how many received 0.3.

Q. What are the BIS numbers for the groups that received remifentanil?

A. Those data -- go ahead.

Q. Is that in Table 1?

A. No. That is going to be in the

Figure 2. I don't know that they report it anywhere else. Let me just look and see.

So, it looks like the data are only going to be in Figure 2. I don't know that they have it anywhere else. But on the top figure of Figure 2, which says BIS, that's where you see the BIS numbers essentially. You can see that they are in the range of -- you know, in the 60's or so. It is hard to tell from that graph exactly where they are, but it is in the same range as described in the preliminary group that they studied.

But also, you don't really see an increase in the BIS over time. It is basically staying pretty flat. It goes down from 96 and stays in the 60's basically.

Q. So, in this study, were the subjects subjected to any noxious stimuli or consciousness check?

A. During the period when they had received midazolam and had been intubated,

for GABA and there is only so much GABA. And when you exhaust that, mechanistically, it will have no further effect. Correct?

A. I am not sure I would characterize that assessment because what you are talking about basically is you are extrapolating from what essentially are some in vitro studies that look at this to the clinical situation. And it's fraught to extrapolate from in vitro studies to this situation.

So, for example, in a typical study if you were looking at a ceiling effect in vitro, you would give huge amounts of the drug over a broad concentration range such that usually you are giving what we would call log doses, log, as in L-O-G. That is, you might be giving one unit of the drug, again in vitro, and then you do a study where you give 10 units and then 100 units and then 1,000 units and so forth. And that is how you can demonstrate an in vitro ceiling

effect.

Now, clinically, in this situation, they have gone from .2 milligrams per kilogram to .3 milligrams per kilogram, and that is only a 50 percent increase basically from .2 to .3. So, although the authors here do bring up the possibility -- they don't mention the ceiling effect herem but they bring up this possibility of saturation. You know, they haven't explored fully the concentration ranges that you would need to be able to make that kind of determination.

Q. So the authors in this paper that you are relying upon raise the issue of midazolam having a ceiling effect. Is that fair?

A. I don't know that they use that term. They say saturation at the benzodiazapine receptor site would account for the small differences on EEG between patients receiving midazolam .2 and .3. Now, they say there were some differences.

They were small. However, again, you would not necessarily be able to detect changes beyond that if you are just going from .2 to .3 milligrams per kilogram.

Q. What is your basis in science or your understanding of this mechanism to opine, if you do, that midazolam, which has this singular mechanism of action with GABA and GABA receptors, that if, by increasing the dose by 50 percent, you get a small additional effect, that that is not due to saturation? Do you have a basis to believe that is not due to saturation of the benzodiazapine receptor sites?

A. Again, I would say that my basis for that is simply that we are looking at what essentially is a dose response effect. That is, we are giving a dose of a drug and we are looking at a response. In this case it is the electroencephalogram. That might be a very shallow dose response so that, you know, it is not flat, it is just very

shallow and you are not going to see much of a change over that concentration range.

Q. And then what is your basis -- do you accept that the benzodiazepines, including midazolam, have this singular mechanism of action; that is, the GABA, that it requires GABA and GABA receptors to be present?

A. Yes. That has been well documented, so I do accept that as the mechanism by which these drugs work.

Q. Okay. And at 0.2 to 0.3, increase of 50 percent, which for a 100-kilogram person would be an increase from 20 to 30 milligrams, they are reporting a very small change. Correct?

A. Yes.

Q. And what is the change in BIS that they are reporting?

A. I am not sure that they reported a change in BIS. In their discussion again they state that -- they said small changes. I don't know -- or "small differences." I don't know what they are

referring to there. I am just reading what they have written there. I have to look at the paper more closely to figure out what small differences are they referring to.

Q. But if you look at the conclusion, it says, "In conclusion, among our patients the average BIS remained greater than 60 with midazolam at 0.2 or 0.3 despite the rapid decrease in plasma concentration."

So, there is no difference -- they are not -- there is no notable difference between .2 and .3 milligrams per kilogram in their conclusion. Correct?

A. They do not identify any differences between those two groups if -- sorry. Maybe I misunderstood your question here.

Q. So, what is your basis to believe that there would be a higher ceiling if you -- well, let me withdraw that.

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Do you have any basis to believe that the failure to observe any notable change in BIS when you increase the dose from what would be the equivalent of 20 milligrams to 30 milligrams for a 100-kilogram person, that that -- do you have any basis to believe that that is not attributable to a saturation of the GABA and GABA receptors?

A. Well, once again, I would say that a change from .2 to .3 is just not a large enough difference to be able to detect what may be some subtle changes that occur, so that to achieve more of an effect, more drug would have to be given. If they had given a much larger dose of the drug, then that would provide maybe some more evidence that there is not much of an effect as you increase the dose.

Just going from .2 to .3 in my mind is not sufficient to state that absolutely there is no change between .2 and .3. You need a broader dosage range in my opinion.

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Q. You may have misunderstood my question but you didn't respond to it.

My question was, do you have a reason to believe that the observation that there was no notable change in BIS values when you increased the dose from 0.2 to 0.3 milligrams per kilogram, or 20 to 30 milligrams per kilogram -- 20 to 30 milligrams in a 100-kilogram person. Do you have any reason to believe that that is not due to saturation of the GABA and GABA receptors?

MR. MANSINGHANI: Object to form.

A. I'd have to think about that. Is there any other reason that -- again, I would say the reason is that it has not -- it is not a sufficient dose difference to be able to rule out basically a subtle effect. So, I don't know how else to answer your question.

Q. Well, the authors of this paper opine that saturation at the benzodiazapine receptor would account for

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why I am going to argue with you about this. I don't know the origin of all this data, but all I know is there are case reports of people who received midazolam by itself and were left unattended and not monitored and they died. So, there may not be a dose listed in this table, but there have been doses of just a few milligrams or 5 milligrams or maybe 10 milligrams that have killed human beings. So, there are doses that kill patients.

Q. And what is the mechanism of death in those cases if you know?

A. It's going to be a combination of unconsciousness, airway obstruction and decreased breathing, respiratory arrest --

Q. Vomiting --

A. That's the usual mechanism.

Q. Are you aware that midazolam is understood to be variable in its effect based on the genetic makeup of the patient?

MR. MANSINGHANI: Object to

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form.

A. There are pharmacokinetic differences basically among individuals for a variety of different drugs, and that holds true for a drug like midazolam as far as I am aware. But as you get into larger and larger doses, you begin to collapse, so to speak, that difference. That is, you still achieve the same end point; you just have to give more drugs to some of these individuals.

Q. But how is the difference explained if you know? Is it based on the identity and the amount of the GABA receptors and the GABA in those individuals as a result of the genetic makeup, or is there a different mechanism that explains the variability, Doctor?

MR. MANSINGHANI: Object to form.

A. I don't know the answer to that question. I know that there are genetic differences. I don't know whether it is related to the makeup of the GABA receptor

or something else.

Q. But if it is related to the makeup of the GABA receptor or the amount of GABA, individuals may also have different ceilings on the effect of midazolam. Correct?

A. I don't know that you can make that extrapolation. So, I am sorry, I am not going to agree with that.

Q. You don't know one way or the other?

A. I do not.

Q. Let's go to the Glass reference, please -- oh, let's go to your report, Doctor, at page 7 -- paragraph 7, rather. There is a graph here.

A. Yes.

Q. You know the graph I am talking about that graphs anesthetic concentration versus percent of patients responding? Do you remember that?

A. Yes, I do.

MR. MANSINGHANI: Pilar, we can see your screen along with the live

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of midazolam for the appendectomy, but I wouldn't need to use a lot of vecuronium.

MR. STRONSKI: Thank you, sir. I think my time is up.

THE WITNESS: Thank you.

MR. MANSINGHANI: I do not have any direct.

THE VIDEOGRAPHER: We are off the record at 7:55 p.m. Eastern Time. This concludes today's testimony given by Dr. Joseph F. Antognini. The total number of media units used was 7 and will be retained by Winter Reporting, a Veritext company.

[TIME NOTED: 7:56 p.m.]

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DR. JOSEPH ANTOGNINI

SUBSCRIBED AND SWORN TO
BEFORE ME THIS_____DAY
OF_____, 2021.

_____
NOTARY PUBLIC

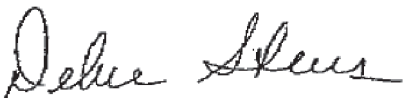
CERTIFICATION

I, DEBRA STEVENS, a Notary Public for and within the State of New York, do hereby certify:

That the witness whose testimony as herein set forth, was duly sworn by me; and that the within transcript is a true record of the remote testimony given by said witness.

I further certify that I am not related to any of the parties to this action by blood or marriage, and that I am in no way interested in the outcome of this matter.

IN WITNESS WHEREOF, I have hereunto set my hand this 9th day of February, 2021.



DEBRA STEVENS, RPR-CRR

WITNESS ERRATA SHEET

CASE NAME: Glossip v. Chandler
DATE OF DEPOSITION: January 28, 2021
WITNESS NAME: DR. JOSEPH ANTOGNINI

PAGE/LINE(S)/ CHANGE REASON
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DR. JOSEPH ANTOGNINI

SUBSCRIBED AND SWORN TO
BEFORE ME THIS_______DAY
OF_________________, 2021.

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NOTARY PUBLIC
MY COMMISSION EXPIRES____________________

Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate. The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

DISCLAIMER: THE FOREGOING FEDERAL PROCEDURE RULES ARE PROVIDED FOR INFORMATIONAL PURPOSES ONLY. THE ABOVE RULES ARE CURRENT AS OF APRIL 1, 2019. PLEASE REFER TO THE APPLICABLE FEDERAL RULES OF CIVIL PROCEDURE FOR UP-TO-DATE INFORMATION.

VERITEXT LEGAL SOLUTIONS
COMPANY CERTIFICATE AND DISCLOSURE STATEMENT

Veritext Legal Solutions represents that the foregoing transcript is a true, correct and complete transcript of the colloquies, questions and answers as submitted by the court reporter. Veritext Legal Solutions further represents that the attached exhibits, if any, are true, correct and complete documents as submitted by the court reporter and/or attorneys in relation to this deposition and that the documents were processed in accordance with our litigation support and production standards.

Veritext Legal Solutions is committed to maintaining the confidentiality of client and witness information, in accordance with the regulations promulgated under the Health Insurance Portability and Accountability Act (HIPAA), as amended with respect to protected health information and the Gramm-Leach-Bliley Act, as amended, with respect to Personally Identifiable Information (PII). Physical transcripts and exhibits are managed under strict facility and personnel access controls. Electronic files of documents are stored in encrypted form and are transmitted in an encrypted fashion to authenticated parties who are permitted to access the material. Our data is hosted in a Tier 4 SSAE 16 certified facility.

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